THE VETERINARY RECORD

FOUNDED BY WILLIAM HUNTING, F.R.C.V.S., 1888

No. 45. NOVEMBER 5, 1949 VOL 61.

PAPERS PRESENTED TO CONGRESS, N.V.M.A.

(*Continued from page 724*)

EXPERIMENTAL RICKETS AND TETANY IN THE FOX AND THE DOG

By

DR. ARNE HELGEBOSTAD AND DR. NILS BOHLER

(OSLO, NORWAY)

The proceedings at this session of Congress, held in the Bishop Partridge Hall at Church House, Westminster, on the morning of Saturday, August 13th, 1949, were presided over by Major HAMILTON KIRK, M.R.C.V.S., of Finchley Road, London.

The CHAIRMAN: We are fortunate this morning in being about to hear a discourse upon the subject of " Rickets and Tetany in the Fox and the Dog " by the two Norwegian essayists, Dr. Helgebostad and Dr. Böhler. I understand, however, that Dr. Böhler, who is on the staff of the Veterinary College at Oslo, is unable to attend. But Dr. Helgebostad is here, and he is the Chief of the Research Station for Furred Animals at Asker in Norway. Both of those gentlemen have extensive expert knowledge of the subject under discussion. I personally read their paper with very great interest because it seems to me a clear exposition of the causes of this important and quite perplexing disease, and I believe it will go far to clear up many of the points which have exercised our minds. I am not asking Dr. Helgebostad to read his paper through, but I shall invite him to the rostrum for a few minutes in order that he may summarise this joint paper or emphasise any particular points which he thinks of importance.

Dr. HELGEBOSTAD expressed his gratification at the opportunity afforded him of participating in the Congress and continued: Investigations concerning the rickets-tetany syndrome in foxes and dogs are rare in veterinary literature. As diseases characterised by nervous symptoms often arise among foxes and dogs in captivity, the authors have considered it to be a matter of importance to investigate under what conditions rickets and tetany will be likely to occur in practice.

Rickets has been induced in fox-cubs and puppies by using different types of rachitogenic diet, *viz.*:

1. A diet poor in phosphorus, but rich in calcium.
2. A diet poor in calcium, but with moderate or high content of phosphorus.

Our experiments have shown that rachitic foxes and dogs are particularly sensitive to rapidly occurring changes in the diet. We believe it to be of interest to mention that spasms and convulsions in connection with sudden alterations of the diet have appeared in animals in which no visible demonstrable symptoms of rickets could be detected. This liability in foxes and dogs ought to receive special attention, and we are of the opinion that cases of tetany in foxes and dogs from the time of weaning to the age of three to four months may be due to a more or less marked rachitic condition, which in the event of changes in the composition of the feed may give rise to outbreaks of tetany.

The CHAIRMAN: I thank you, Dr. Helgebostad. The discussion will be opened by Professor Alastair Worden, who is chief biochemist and nutritionist at the Animal Health Trust at St. Ives; and the co-opener is Mr. A. A. Wilson, until recently studying biochemistry at Liverpool University. [Following is the paper.]

Experimental Rickets and Tetany in the Fox and the Dog *

The occurrence of rickets and tetany in foxes is seldom mentioned in the literature. It is a fact, however, that diseases attended by nervous symptoms often occur in foxes. These diseases may be infectious, parasitic, toxic or alimentary in nature.

Tetany in combination with rickets has been observed in children and in some of our domestic animals and it was this syndrome we wished to study more fully through experiments on foxes and dogs. Rickets was produced in fox-cubs and in puppies by the use of two different forms of diet.

One diet was poor in phosphorus, but rich in calcium.

The other was poor in calcium and contained a moderate or large excess of phosphorus.

In the first-mentioned group we have adopted Steenbock and Black's rachitogenic diet No. 2965 for rats; in the other group either Harrison and Mellanby's rachitic diet for dogs or a diet consisting of boneless meat with addition of Na_2HPO_4. Both these types of diet have led to rickets in the animals.

It is maintained by several investigators that a diet with low calcium and high phosphorus content will not lead to rickets, but to pseudorickets or an osteoporotic condition. It is stated that lack of calcium in the feed has an inhibitive effect on the growth and therefore produces unfavourable conditions for development of rickets. In our experiments several animals, in spite of poor growth, showed unmistakable symptoms of rickets. Practical experience shows, however, that animals in vigorous growth are particularly liable to rickets.

The development of rickets invariably is associated with the period of growth, and it is generally believed that hereditary pre-disposition, bad hygienic conditions, lack of sunshine, of fresh air and exercise play an important role in the development of the disease. In our experiments we have paid special attention to the alimentary factors and the effects of light.

Abnormal ossification of the bones may arise from deficient supply both of calcium and phosphorus, but also from the exclusive administration of phosphorus or calcium.

* Presented to the Sixty-seventh Annual General Meeting and Congress of the National Veterinary Medical Association, in London, August 13th, 1949.

Vitamin D has a favourable effect on the utilisation of calcium and phosphorus in the organism. Vitamin D is usually assigned greater importance in infantile rickets in man than in the different forms of the disease in animals.

An essential cause of rickets is further to be found in factors which have the effect of preventing absorption of calcium and phosphorus from the intestines by forming almost insoluble compounds of lime and phosphorus such as, for instance, phytin.

Harrison and Mellanby have made a thorough study of the properties of phytin. They find that phytic acid and its sodium salts have a highly rachitogenic action. Phytic acid forms with calcium the almost insoluble calcium phytate, which is not absorbed. The same authors maintain that the rachitogenic action must be due to the power of the phytin to bind far larger quantities of calcium than the cereals themselves contain.

The rachitogenic action of the different cereals cannot be estimated solely from their content of calcium, phosphorus and phytin. There are several cereals which do not produce a predisposition to rickets, in spite of their high content of phytin. In these cases the cereal contains the enzyme phytase, which splits the phytin into easily absorbable phosphorus compounds. Barley, rye and wheat contain phytase and thus have a far less marked rachitogenic effect than oats and maize, in which no phytase is available and phytin may constitute from 80-90 per cent. of the total content of phosphorus.

Splitting of phytin in the alimentary tract by aid of the bacterial flora in the intestine has previously been assigned little importance. American investigators (Phillips, Spitzer), however, have recently claimed to be able to detect a special intestinal phytase in rats.

OUR OWN EXPERIMENTS

Low-phosphorus Rickets

To the group with low content of phosphorus there was given a basal diet corresponding to Steenbock and Black's rachitogenic diet for rats. Its composition was as follows:

74.5 g. of bruised maize	... (74.5 per cent.)
20.0 g. of wheat gluten	... (20.0 per cent.)
4.0 g. of calcium carbonate	... (4.0 per cent.)
0.5 g. of dried beer yeast (non-irradiated)	... (0.5 per cent.)
1.0 g. of common salt	... (1.0 per cent.)

In addition to this ration each animal was given daily 12.5 to 20 g. of lean boneless veal to stimulate the appetite, as well as 0.5 mg of aneurin and 0.5 mg of carotene. The animals consumed from 100 to 200 g. of dry-feed per day. This diet is especially deficient with respect to phosphorus, but contains a large excess of calcium $Ca/P > 5.4$. Most of the phosphorus is present in the form of phytin phosphorus and therefore cannot be utilised by the organism, since maize is almost free from the enzyme phytase.

The subjects of experiment included 19 fox-cubs and two puppies. They were kept in the dark and placed on raised netting. The mothers of these animals were fed a diet poor in vitamin D during the preceding period of gestation and suckling. The experiments were started immediately after the young animals had been weaned.

In the course of three or four weeks the first symptoms of rickets were noted. The animals showed incipient enlargement of the joints and abnormal position of the legs. By degrees they sank down on the knees, hams and toe-joints. They seemed small of growth and finally came to have a completely plantigrade gait.

The blood showed normal or increased calcium levels of about 10-12 mg per cent. while the inorganic serum phosphorus in the course of the experiment fell from about 8 mg per cent. to between 3 and 5 mg per cent. Nervous symptoms were not observed as long as the animals were kept on the rachitogenic diet. Nor were such symptoms to be expected, seeing that the animals were receiving a supplement of lime in the feed, and the serum calcium was normal or even increased. If a change was made in the diet, however, and the animals were given easily absorbable phosphorus compounds, in boneless meat or fish-fillets, they promptly re-acted with tetany in the course of from two to five hours after the change. This circumstance will be discussed more fully in connection with tetany.

X-ray pictures of the animals in the low-phosphorus group revealed in many cases pronounced rachitic changes with considerable thickening of the epiphyseal cartilage in the extremities.

Low-calcium Rickets

In these experiments 12 silver-fox cubs and two puppies were employed. The animals came from the same litter as those in the preceding experiments and were reared under the same conditions. Six fox-cubs and and one puppy received Harrison and Mellanby's rachitogenic diet of the following composition:

Dried milk (skim milk)	20 g.–30 g.
Oatmeal from whole oats	65 g.–160 g.
Baker's yeast	3.5 g.–5 g.
Peanut oil	8 g.–12 g.
Cabbage	14 g.–20 g.
Veal	14 g.–20 g.
Common salt	1.2 g.–2.3 g.

The remaining foxes received a diet consisting of boneless meat or meat $+Na_2HPO_4$. In addition, they got a daily supplement of ½ mg aneurin and ½ mg carotene.

Harrison and Mellanby's diet is distinctly poor in calcium $Ca/P<0,85$. The phosphorus is present mainly in the form of phytin and will therefore bind a large part of the lime, thus the amount of calcium actually disposable is far less than is revealed by the analysis. The diet with boneless meat or meat with addition of Na_2HPO_4 has nevertheless a decidedly uneven calcium/phosphorus balance. Even though this diet does not contain phytin, it leads to the same histopathological changes as Harrison and Mellanby's diet.

In the low-calcium group symptoms of rickets appeared after 6 to 8 weeks' feeding. The clinical picture differed sharply from that seen in animals on diet poor in phosphorus. The enlargements around the joints were not so conspicuous and developed distinctly more slowly in this group. A very prominent symptom was increasing soreness in the limbs. After three or four weeks the animals already showed a cautious gait and kept very quiet. Later on the movements assumed a spastic appearance and we noted increased tonus in the muscles. This spasmophilia was due to low content of calcium in the blood, with values right down to 6 or 7 mg per cent. At this time the animals were nervous, they reacted quickly to unaccustomed sounds and the reflex irritability was increased distinctly. Direct attacks of cramps were seldom noted. As mentioned previously, the picture of nervousness which here gradually developed, was not observed in the phosphorus group. The X-ray pictures also differed in the two groups.

While in the low-P group we found a typical rachitic

metaphysis with considerable increase of the epiphyseal cartilage, the roentgenograms in the low-Ca group showed moderate thickenings of the epiphyseal cartilage, ill-defined bone-contours and effacement of the bone-structure itself.

The changes seen in the bones of the dog in the low-Ca group were almost identical with those found in the bones of the animals in the low-P group. The roentgenograms showed very broad metaphyses. Otherwise the dog behaved similarly to the foxes on the same diet, but showed considerably better growth.

The subtetanic condition in the dog repeatedly developed into manifest tetany with cramps and compulsive movements. The nervous symptoms set in by degrees and were most pronounced after the experiments had been pursued for eight to ten weeks.

In deficiency states, especially those relating to mineral metabolism, very valuable information can be obtained through a careful evaluation of the changes in the sustaining tissues (bone and dentine).

The literature of the subject records that the rachitic changes in the skeleton have been subjected to exceedingly comprehensive investigations in respect to both human and veterinary pathology. The diagnosis of distinct rachitic changes therefore should not present any particular difficulties. Nevertheless, we have attached great importance to an exact definition of rickets as a pathological-anatomical concept. In the first place, it was important to be able to make a certain differential diagnosis respecting skeletal anomalies of different nature which not infrequently may show changes similar to those seen in rickets. Secondly, it was of especial interest to see whether the changes produced in the two main experimental groups would show fundamentally important deviations. In the judgment of the skeletal changes we are essentially in agreement with the views advanced by Wilton in several publications. Here it is especially emphasized that rickets develops as a result of some injurious influence on osteoblasts and dentinoblasts. This injurious action entails an inhibition of differentiation in the cells. Such inhibition leads to an abnormally increased proliferation of the immature osteoblasts, while at the same time the matrix is formed in relatively large quantity, but acquires an abnormal, irregular structure with uneven and defective mineralisation.

In the low-P group there was found in most cases a very characteristic rachitic picture, both microscopical and histological. Some few animals showed inconspicuous skeletal changes. All these animals previously had shown a marked tendency to tetany and died after a very short experimental period from the attacks of convulsions induced. The histological symptoms can be briefly summed up as follows: Broad layers of epiphyseal cartilage, with jagged, uneven transition to the defective primary spongiosa. The bone-trabeculæ are thick and plump, with considerable osteoid deposits. The osteoblastema is disordered, highly cellular, with many multinucleated syncytia. The marrow cavities were often completely filled with proliferation cells resembling fibroblasts. As a rule great vascular injection and considerable abnormal vascularisation of the cartilage were seen. The arrangement of the trabeculæ was highly irregular.

In the low-Ca group we were prepared beforehand to find that the changes would be somewhat different, the more so because the X-ray examinations revealed somewhat divergent pictures. Macroscopically there were generally found marked changes, the long bones being thick and deformed, soft and flexible. Considerable periarticular enlargements and subluxations were seen in many of the joints of the extremities. The histological examinations yielded a somewhat surprising result, in as much as was found also in this group characteristic and distinctly hyperplastic rachitis in practically all cases. We could in no case detect changes that would justify the use of the designation osteoporosis or porotic rachitis. In these foxes examination of the teeth was also carried out to a large extent and the structure of the teeth was undoubtedly of rachitic character, as there were found a greatly proliferating, sometimes somewhat disordered odontoblastema, abnormally broad predentin layers and irregular, globular calcification in the dentine.

The central nervous system and the peripheral nerves showed normal structure. The conclusion to be drawn from the pathological-anatomical findings will be, then, that the two forms of diet, which are seen to have differed greatly in their content of minerals, have produced entirely analogous and characteristic rachitic changes in the skeleton and teeth. The apparent difference which was noted on X-ray examination we cannot conceive to be anything other than a difference of degree in the mineralisation, without any connection with osteoporotic changes.

Tetany

The object of the present investigation of rickets has been, especially, to obtain a better understanding of the rickets-tetany syndrome in foxes and dogs. To secure a more thorough study of the tetany symptoms in themselves, we have induced the disease experimentally in different ways, partly by parathyroidectomy and partly by intravenous injection of various salts, such as sodium phosphates, sodium bicarbonate and guanidine chloride. We shall not give any detailed description of the experiments, but merely mention that the tetanic symptoms which appeared, were practically the same in all cases.

Total parathyroidectomy leads in the course of a few days to fully developed tetany in foxes, when the animals are put on a diet poor in calcium and free from vitamin D, such as lean meat.

Experimental tetany is of minor interest in veterinary practice. Our experiments in this field have been exclusively dictated by the desire to obtain a knowledge of the forms in which the disease manifests itself.

It appears from the detailed description of the feeding experiments that both spasmophilia and tetany arise in close connection with rickets.

Spasmophilia and Tetany in Cases of Low-phosphorus Rickets

If the animals are kept on a decidedly low-phosphorus diet of the type adopted in the experiments described, the skeletal symptoms will become more marked by degrees and finally we will have the typical picture of rickets. Apart from the ordinary clinical symptoms of this disease, the foxes and dogs show no signs either of spasmophilia or tetany.

The rachitic animals are especially sensitive towards changes of diet when they are given easily absorbable phosphorus compounds. If they receive a meal of boneless meat instead of the rachitogenic diet, they will have manifest tetany within a few hours. They become restless and stiff in their movements. In the course of from two to five hours there arise clonic and tonic spasms. The cubs lie on one side in opisthotonos with violent movements of the limbs. The attacks of convulsions last from a few seconds to a couple of minutes. The cubs continue to lie almost in

a state of coma, either with entirely relaxed muscles or with increased tonus. In other cubs under the same circumstances a more paretic condition arises after the change of diet. *They may also get attacks of convulsions.*

Examination of blood serum during tetany showed pronounced hypocalcemia, with Ca values of 6 to 7 mg per cent. The fall in serum calcium was accompanied by a rise in content of inorganic phosphorus in the blood from 3–4 mg per cent. up to 7–8 mg per cent.

On treatment with calcium injections the nervous symptoms rapidly disappeared.

We also investigated the question of how the rachitic fox-cubs and puppies generally reacted to changes in the feed. We tried rations consisting of whole fish, fish-fillets or mixtures of meat or fish with meal, dried milk, potatoes, etc. We selected for preference food combinations which were relatively rich in phosphorus and comparatively poor in calcium.

The effect of a sudden change of the diet was very marked. Shortly after the addition of meat or fish the animals reacted with nervous symptoms. As a rule convulsions set in from two to five hours after the change of feed.

In this manner it was possible at any time to evoke symptoms of tetany—merely by a temporary change in the composition of the feed. The experiments were repeated again and again with the same animals, but also with other animals. The results were always the same. An alteration in the food given to the rachitic animals, always resulted in nervous symptoms, with more or less pronounced tendency to spasms.

Nervous symptoms likewise may arise when rachitic cubs or puppies are subjected to hunger for a couple of days. Also in this case the disease is revealed by a fall in the blood calcium content, while on the other hand, the serum phosphorus shows higher values than before.

We have instituted experiments, further, *with foxes on non-rachitogenic diets having a low content of phosphorus and a high content of calcium.* Here it was not possible to detect any particular alteration in the blood calcium level after change of diet.

This finding has been confirmed through experiments *on full-grown animals.* Adult foxes that had been kept on rachitogenic diet for a considerable time *showed no nervous symptoms after the change of feed.* It is, however, of special interest to note that in several cases, where we made a sudden change in the feed by giving meat, fish, etc., we obtained pronounced *tetanic symptoms,* such as convulsions, *in two or three months old silver-fox cubs which had been on a rachitogenic diet for a relatively short time* (14 days)—*without any definite symptoms of rickets having previously been detected.*

We have established that this form of alimentary tetany in rachitic animals is due to an action of phosphorus compounds. In order to investigate this point we gave, instead of meat, fish, etc., small quantities of Na_2HPO_4 (100 mg.) per os to the same animals. A good supply of pure phosphates to rachitic cubs repeatedly led to tetany, characterized by low calcium and high phosphorus content in the blood, as well as by pronounced nervous symptoms.

Spasmophilia and Tetany in conjunction with Low-calcium Rickets

From our experiments it appears that rickets can be induced in young foxes and dogs also by means of low-calcium diets. Meanwhile it took a comparatively long time (6—8 weeks) before the disease could be detected clinically. The nervous symptoms came on by degrees and did not appear until after an experimental period of 6—8 weeks.

When the nervous symptoms set in, the animals constantly showed a neuromuscular irritability, with definite symptoms of spasmophilia. The serum calcium values were subnormal, from 6 to 8 mg per cent, while the content of inorganic phosphorus in serum was moderately reduced, having values from 4 to 5 mg per cent. The cubs were easily startled (on hearing unaccustomed noises), and on palpation we noted a distinct increase of muscular tonus. Their gait might be stiff and constrained. At this stage the animals were in a state of latent tetany (spasmophilic tendency), without necessarily being attacked by convulsions.

Attempts to induce tetany by a sudden change of the Ca/P ratio in the feed did not lead to aggravation of the nervous symptoms.

The relative rarity of attacks of spasms in the low-calcium group is explained as being due to *the animals having gradually become accustomed to that form of hypocalcaemia.* The same phenomenon was observed in the case of the parathyroidectomised cubs. When they were getting normal feed with a sufficiency of calcium and vitamin D, they might remain apparently well for months, with hypocalcæmia corresponding to 6—7 mg per cent. Ca.

On the other hand a dog which underwent the same experiments showed constrained movements after two months and sustained repeated tetanic attacks.

Fox-cubs which were given boneless meat with addition of sodium phosphate and which had abundant access to sunshine failed to develop spasmophlia. The blood calcium was normal during the whole period of experiment. In full-grown foxes subjected to the same experiment tetany likewise failed to develop, even if they were kept in a dark place.

A more detailed description of this work is given by Fr. Ender, A. Helgebostad and N. Bohler, *Studies in Experimental Rickets and Tetany in Foxes. Oslo* 1949 (in press).

Discussion

Professor Alastair Worden, M.A. (Cantab.), B.Sc. (Lond.), M.R.C.V.S., F.R.I.C. (Animal Health Trust Station, St. Ives, Hunts.): I should like to congratulate our Norwegian colleagues upon their contribution and to say how glad I am that we have been promised a more detailed account of their experiments. I do not wish to take up the time of the meeting by asking for clarification of certain of the biochemical and histological points, as these will doubtless be amply discussed in the forthcoming monograph. Rickets and rachitic-like skeletal disorders have been the subject of considerable study, even within the carnivora, and, as will be recalled by those who heard or read the papers based upon the detailed lectures given by Sir Arnold Theiler in 1933, there has been confusion over the interpretation of the pathological lesions in the dog. Theiler, incidentally, was satisfied that a true rickets occurred in the dog (in which it was, in his opinion, primarily an avitaminosis) but he considered that osteodystrophia fibrosa also could occur in this species, although less commonly, and that clinically the two conditions could be confused. He appears to have accepted the experimental reproduction of osteodystrophia fibrosa in the dog by Weber and Beck, but apparently he felt that their data did not completely establish their view that a dual deficiency of calcium and vitamin D was responsible.

In the time at our disposal, and in view of the necessarily abbreviated account before us, and of the emphasis that the authors have laid upon the nervous manifestations, I would suggest that to-day's discussion be concentrated upon clinical lines. I hope, Mr. Chairman, that we shall have the benefit of your very considerable experience of small-animal practice. The subject of nervous disorder in the dog (and, in appropriate practices, in the fox) is a very topical one and any light that may be shed upon

the relationship of diet to abnormal nervous behaviour is of distinct veterinary interest. It is, of course, unfortunate from the standpoint of most of those present to-day that so few dogs were employed in the experiments reported, although there were doubtless serious practical reasons for this omission. I do not think, from published data and in view of the discrepancies between different dogs in their susceptibility to rickets, that the use of one or two dogs can be said to have afforded adequate controls to the foxes. Another adverse criticism, although it may be unjustified, concerns the lack of any references to worming or to precautions against possible infection. In his studies at Wisconsin upon diet in relation to canine hysteria, my colleague, H. B. Parry, was most careful to try to exclude intestinal helminthiasis and infective agents, both of which may be associated with the manifestation of nervous symptoms.

The relationship between a low-calcium diet and what has been termed "pre-rachitic tetany" in children, in piglets, and in the young of other species has of course been realised for very many years (for references see J. Pickup, 1942, *Veterinary Record*, **54**, 97-98 and A. T. Shohl's *Mineral Metabolism*, New York, 1939) and in the case of piglets was described by Hutyra and Marek in editions of their well-known textbook published before World War I. We now have evidence that in fox-cubs receiving a rachitogenic diet low in phosphorus, tetanic convulsions may readily be induced, even in the pre-rachitic stages, by the addition of meat or fish, or even by the administration of inorganic phosphates. I assume from the wording of their paper that the authors are satisfied that the same mechanism operates in puppies similarly fed, and indeed a high phosphate tetany was produced in dogs by Salvesen and his colleagues as long ago as 1924, in their case also by oral dosage. Shohl has drawn attention to the possible association of deranged mineral metabolism with epilepsy, and while I certainly do not wish to press the analogy too far, I think it is well worth our while to recall the association of sudden changes of diet to hysteria and epileptiform convulsions in dogs. It was common experience in the North London area prior to the war that hysteria and even "grand mal" convulsions in susceptible dogs could result from, and be ended by, sudden changes in the diet. From the experimental work already referred to, Parry believed that certain diets could be regarded as "hysteria-inducing," *i.e.*, capable of setting up a "hysteria-prone"-condition, whereas others were to be classified as either "hysteria-precipitating" or "hysteria-depressing," *i.e.*, those diets a change to which would, respectively, precipitate or reduce the risk of motor episodes in the dog already rendered prone to hysteria. The whole subject of nervous manifestations in relation to changes of diet is one concerning which practitioners have, I feel sure, as yet a great deal to tell us. So-called "teething fits" are not without their importance in veterinary practice, if only from the standpoint of differential diagnosis; indeed, in French textbooks, in which they are known as "rage dentaire," care is taken to differentiate them from rabies, while in this country, certainly in the area in which I saw practice during the 1930s, the initial convulsion that often heralded distemper (or at least, a condition that in those days responded dramatically to distemper antiserum) was frequently dubbed a "teething fit" by the owner. Are teething fits induced or precipitated, at the time of active tooth-growth and consequent drain upon calcium metabolism, by certain types of diet?

Dr. Helgebostad and Dr. Böhler have made little reference in to-day's paper to vitamin D except in their brief notes upon parathyroidectomised foxes. They appear, in fact, to have excluded it as a complicating factor and to have avoided light. I should like to enquire whether, in experiments not reported to-day, they were able to prevent the various manifestations by administering vitamin D, or whether they are satisfied that such would be the case in view of the ability of this vitamin to overcome the effects of a disproportionate Ca/P ratio. I should like to enquire, further, whether vitamin D was used curatively in any of their experiments and, if so, what were the immediate effects. Spasmophila or infantile tetany in the human subject, when associated with a deficiency of vitamin D, is said to indicate the beginning of healing. To quote H. R. Rosenburg (*Chemistry and Physiology of the Vitamins*, New York, 1942, p. 425) "Etiologically, vitamin D cures the tetany of children but not symptomatically, or only after a considerable length of time." Since the work of Mellanby we have, I believe, been conscious of the need of vitamin D by the growing puppy; and indeed one modern version of the once maligned condition powder contains, among other nutritional supplements, a considerable quantity of calciferol (vitamin D_3). Yet, as mentioned above, the vitamin D (or at least the vitamin D_3) requirements of different dogs vary widely. A. F. Morgan (*N. Amer. Vet.* (1904). **21.** 462) has shown that large breeds such as the Great Dane, Setter, Airedale and Alsatian need relatively far more vitamin D_3—even ten times or more on a bodyweight basis—than smaller breeds employed. I believe it to be clinical experience in this country that the Great Dane especially is liable to rickets. I should like to enquire what breed or cross breed of dog was employed by Dr. Helgebostad and Dr. Böhler, and whether—if it was not a very large breed or type—they might not have expected more dramatic results from the use of such animals.

I should like to enquire, further, what were the sexes of the experimental cubs and puppies. Male puppies are said to be more susceptible than female to rickets—it would be useful to have practitioners' views upon this—and one wonders whether this, if it holds true generally, is a growth effect *per se* or whether it is connected with hormonal metabolism. I believe that in East Anglia some years ago, Blakemore found interesting sex differences in the susceptibility of cattle to aphosphorosis.

Finally, if it is permissible in what I was originally informed was to be a discussion on rickets and tetany in carnivora, I should like to make a point about cats. Cases of rickets in Felidae have been reported from zoological gardens (*e.g.*, H. Fox, *Diseases in Captive Wild Mammals and Birds, Philadelphia*, 1923, p. 358) but if I may quote from a letter from Dr. J. R. M. Innes ". . . it is an extraordinary thing, but rickets, or indeed any rachitic-like skeletal disorder, has never been recorded in the domestic cat, nor has the cat ever been used in experimental work dealing with Ca/P metabolism and vitamin D." Has anyone here ever seen what he or she believed to be rickets or pre-rachitic tetany in a cat, and if so, what were the circumstances?

I must apologise if I have not limited my remarks to discussion of the experiments before us. I feel, however, that those of us who are interested in the more academic aspect of Dr. Helgebostad's and Dr. Böhler's work can obtain the necessary information from their subsequent detailed publication, and that it would be neither fair nor helpful to begin an intensive discussion upon the summarised version before us. On the other hand, their paper raises or recalls many points of interest to practitioners, and I trust that we may receive from the latter valuable practical observations upon rickets and tetany as encountered in small-animal practice.

Major A. A. Wilson, M.V.Sc., M.R.C.V.S., of Southport, Lancs., who was the second opener of the discussion, said: It is a great pleasure to congratulate Drs. Helgebostad and Böhler on their very interesting paper and I am very pleased to be one of the openers of the discussion. I will confine myself to a few general remarks on the subject of rickets and tetany in dogs and to asking a few questions of the authors.

In infants, rickets is usually the result of the deprivation of vitamin D, but in rats and dogs it has been shown that provided that the ratio of calcium to phosphorus in the diet is normal, rickets will not develop. Much of the importance attributed to the Ca/P ratio would appear to be related to its effect on solubility. A high Ca/P ratio definitely lowers the phosphate absorption, though a low ratio does not hinder calcium absorption. Thus on a low phosphorus diet there is not only the effect of the absolute shortage of phosphorus but also the effect of the high Ca/P ratio, tending to decrease the amount of phosphorus which the animal receives. Among other factors influencing the absorption of these two important elements are the acidity of the intestinal contents. J. H. Jones. (1942.) (*J. Biol. Chem.* **142.** 557) showed that lard, oleic acid and vitamin D increased the acidity of the small intestine while aluminium stearate, which lowered the acidity, produced severe rickets. McCance, Lehman and their co-workers have shown that amino acids will increase the solubility of calcium salts and that this effect is also seen with a meat diet, so that meat, though of relatively low Ca content, can be quite a good source of this element as far as the body is concerned.

As I am not in practice I cannot speak from personal experience

but as far as I can judge by the observation of dogs which one sees in the streets, etc., severe rickets is not common in this country. If this is so—and some of our colleagues in practice will no doubt be able to confirm or deny it—it is a rather surprising finding when one considers that large numbers of dogs are fed on what can only be called unnatural diets, in which the Ca/P ratio must be very different from that found in the natural diet of the carnivorous animal, which is meat with the bone. One can only assume that either the dog is not very sensitive to rickets or that sufficient vitamin D is produced in the skin to counter-effect the diet.

Linton has stated that the commonest form of rickets seen in dogs is the low phosphorus form. Would the authors agree with this statement? On a meat diet this would appear to be an unusual finding, particularly as the authors state that they produced low calcium rickets on a diet of boneless meat with or without sodium phosphate. One can only assume that the low phosphorus rickets is due to the habit of feeding dogs on semi-vegetarian diets in which some of the phosphorus will be unavailable owing to its being in the form of phytin. In connection with this point I should like to ask the authors whether they found any difference between those animals in which low calcium rickets was produced by means of Harrison and Mellanby's diet and the rickets produced by means of the boneless meat plus disodium phosphate?

In typical rickets in children there is a lowering of the blood phosphate and an increase in blood phosphatase while the serum calcium remains normal. The authors report a low serum inorganic phosphate in low P rickets; was this also seen in the low Ca rickets in which there was a low serum Ca and was the blood phosphatase changed in either of these forms?

Tetany or spasmophilia are not common findings in infantile rickets though tetany is sometimes seen following the administration of curative doses of vitamin D. It has been suggested that this is due to the rapid action of the vitamin in promoting the deposition of bone salts, as a result of which the calcium is removed from the blood and tetany follows. The authors have reported that the administration of phosphate to their animals with a low P diet resulted in the production of tetany after a few hours, with a fall in serum calcium and a rise in serum phosphate. The mechanism of the production of this tetany would appear to be the same as that which produces tetany in rachitic infants following the administration of vitamin D. That is, there is a removal of calcium from the blood in order that it may be deposited in the bone, and as calcium removed will be mainly calcium ions, tetany will follow. In other words, the solubility product of calcium and phosphate rises to a level which results in precipitation of bone salt. It would be of interest to know whether the authors found any improvement in the rickets following the administration of the phosphate, and also how long the effect of a single dose lasted. The fact that tetany did not follow change of diet in full grown animals kept on a rachitogenic diet supports the view that the fall in serum calcium is due to its being deposited in the bone of the young animals on rachitogenic diets.

The relative rarity of tetanic attacks in the cubs on the low calcium diet is in accord with observations on rats in which it is stated that low calcium diets do not produce tetany but rather muscle weakness. However, the authors have stated that there was an increase in nervous irritability and that the pups or cubs were easily startled. The authors' suggestion that the relative rarity of tetanic attacks, in the low calcium group and in parthyroidectomised animals receiving calcium and vitamin D, is due to the animals having become accustomed to the hypocalcaemia, is of considerable interest. This "conditioning" to low serum calcium could be explained in several ways.

1. The nervous system of the young dog or fox in the growing stage is more labile than that of the adult and can undergo modification.

2. As it is concentration of ionised calcium which is responsible for the control nervous tissue there may be a change in the relative proportions of ionised and bound calcium, so that, although the total serum calcium is low, the ionised calcium has not fallen to the same extent, and the level is still too high for tetany, though low enough to produce increased irritability.

3. Nervous irritability is known to be a function of electrolyte balance, so there may be compensatory changes in other ions. In connection with this point it seems to me that we tend in many matters to harp on single factors as causes whereas we ought not to forget that balance between a number of factors may be of more importance.

4. It may be that in the production of tetany the absolute level of serum calcium is not the important factor, but that tetany is the result of a rapid fall, that is, a rapid change of concentration. However, the fact that an adult dog which underwent similar treatment showed repeated tetanic attacks after two months, does suggest that the absolute level of serum calcium is important.

I should be very interested to hear the authors' opinion as to the relation of this conditioning to hypocalcaemia.

Before leaving the subject of tetany in rachitic animals I should like to ask the authors if they have had any experience in the administration of parathormone to rachitic animals. It is assumed that the increase in serum calcium following the administration of this hormone is due to the withdrawal of calcium from the bones; it would be of interest, therefore, to know whether this occurs in rachitic animals and if so, if the rickets is exacerbated in any way.

While not strictly related to the subject of the paper under discussion, the condition of eclampsia in bitches is possibly connected with the production of tetany in dogs. It has been stated that this condition can be treated successfully with injections of calcium gluconate, which seems to suggest that the condition is one connected with a low level of serum calcium. Particularly towards the end of pregnancy there is a considerable drain on the body of the bitch in respect of calcium and phosphorus, not only for the production of bone but also, particularly in the case of phosphorus, for the formation of other important body constituents. Do the authors think that it would be a reasonable hypothesis that, as a result of pregnancy, particularly in the case of animals unnaturally fed, the bitch may be in a state similar to that which they induced in the three months' old fox cubs so that a sudden change of diet would produce tetanic symptoms? Another possible explanation would be that the changes in hormone balance, which occur at whelping, react back on the pituitary gland and as a result the production of parathyrotropic homone is depressed and the parathormone level falls, followed by the calcium level.

Before concluding, there are one or two random questions in connection with bone and calcium metabolism which I should like to ask the authors. Firstly, are vitamins D_2 and D_3 equally effective in the prevention of rickets in carnivores? Secondly, have the authors any experience of concurrent vitamin A and vitamin D deficiency, and if so, what effects, if any, have they on the development of the nervous symptoms? It is known that vitamin A plays a very considerable part in the formation of bone in that it appears to be responsible for the shaping or moulding of the bone and its texture. It appears to produce its effects by controlling the organisation of the osteoblasts and the osteoclasts, though it has been suggested that its effects are mainly osteoclastic in which case it would seem possible that it would help to maintain the serum calcium level. Though it is generally assumed that the nervous symptoms seen in vitamin A deficiency are secondary to the changes in bone, *i.e.*, due to pressure of overgrown bone, it is possible that vitamin A has a direct effect on nervous tissue.

From the work that has been reported to us this morning I think it is permissible to draw the following conclusions.

1. In canines, rickets, however produced, is a constant histopathological entity though the mode of production will influence the type of concomitant nervous symptoms shown.

2. That in young animals at least, the occurrence of a tetanic attack is more dependent on a change in serum calcium than upon the absolute level.

3. Though this is an excursion into the realm of the obvious, that the more natural the diet of the animal, the better for it. By this I mean that it should be like the diet of the carnivore in the natural state, that is, meat with the bone, in that in such a diet it receives plenty of both calcium and phosphorus while the effect of the amino acids in the meat in increasing the absorption of Ca will help to maintain a proper Ca/P ratio.

In conclusion I should like to express again my thanks and congratulations to Drs. Helgebostad and Böhler for their very interesting paper.

General Discussion

Dr. Mark L. Morris (United States): He had taken no notes, and he would simply speak extemporaneously, so they would pardon him if his remarks were rambling. He had been very much interested in listening to the discussion that morning on the production of experimental rickets in the fox and dog. This was a problem in which they had been interested for about 20 years, and he had been a keen observer of the work in the States. He happened to be personally acquainted with a number of the people mentioned in their discussion that morning—Morgan, Phillips and others—and he had had the privilege of listening to their discussions at many of their American meetings.

His deduction from that morning's discussion would be that the problem perhaps needed further clarification and definition. In other words, what did they mean exactly by rickets? Were they going to define it as that disease which occurred in animals or man due to avitaminosis D, or were they going to call rickets the disease which might occur in bony tissue due to some disturbance in the mineral balance? If they were going to limit their discussion to the disease produced by a lack of vitamin D, then might he suggest to those working with the dog in the future that they used great care to balance the basal diet, so that all essential nutrients were present in proper proportion to nourish the dog adequately. And might he further warn them not to accept diets which had been used in rat studies, as they knew that the biological requirements of the rat and the dog were quite different. The basal diets used in those studies were interesting, but, before he would care to pass judgment, he would want considerably more information concerning chemical composition, the distribution of the essential vitamins, the biological value of the proteins, and the growth curves of the animals employed, because he feared the authors might be dealing with a complex rather than with a specific entity.

He would recite briefly such work as they had done on rickets in dogs, and he might say this was a field in which they had the least experience. He had never seen, however, any nervous manifestations in dogs associated with what they defined as true rickets. One could produce a tetany or nervous manifestation in the dog with hypocalcaemia, and disturbances in the calcium-phosphorus balance of the blood, but he had never seen it in true rachitis due to the absence of vitamin D in an otherwise normal diet. Their dogs were fed what they considered normal diets and grew well, and he thought that in studying rickets the animal should grow well, because vitamin D should not interfere greatly with growth. However, if the protein, vitamin A, or especially factors of the vitamin B complex, were absent, the growth of the foxes or puppies might be interfered with, and the interpretation of one's finding would be difficult.

One other point. He thought he would be very careful, in working with dogs, not to depend upon brewers' yeast as a total source of the B factor. There was a wide variation in the B components of yeast, and they did not in every instance supply all the factors that were needed by the dog, as some of those factors were contained in liver and could only be obtained therefrom.

The Chairman, having expressed indebtedness to Dr. Morris and invited further contributions, without response, said that personally, he could not lay claim to an intensive study of this disease, rickets. He had a busy practice to look after. If a case of rickets came in, one gave appropriate treatment and forgot all about it. But he had noticed, over the years, one or two funny things about rickets. He thought it was generally appreciated or believed that the children of wealthy people were more prone to rickets than, or at least as prone as, the children of poor people, and it was, he believed, because the parents were so anxious to fill them up with anti-rachitic foods and drugs, that they actually conduced to rickets.

Now he would tell them of a case where, perhaps some 20 years ago, he was asked by a breeder of Irish Setters to go down to his kennels near Woking because he had what he called an outbreak of rickets. He was a big breeder, a breeder in a large way. He spent no end of money—and money was no object at all. He had marvellous kennels, but for the previous three years every bitch and every puppy was subject to rickets. Well, he went down there thinking, "Oh, this is easy." He suggested this, that and the other. The breeder said, "Oh! We have done that"—he left him nothing to tell him at all. He (Major Kirk) was still too inexperienced about rickets at the time to see just what was going on. The breeder had used all the proper foods, gave fresh and dried milk, condensed milk, cod liver oil, calcium, casol, oatmeal, fats, yeast, the best beef and vitamins A and D, and the rickets went on and on. He was unable really to help him, and his client was thoroughly dissatisfied. Some time after that he heard through a mutual friend that this man had cut out everything—cut out the lot and just fed these animals on ordinary meat, a little milk, and dog biscuits, and his rickets went. Now that was an eye-opener. This breeder was giving all the anti-rachitic drugs and as soon as he stopped them the kennel was a clean kennel. He said he did that with no knowledge, it was a pure accident. He just gave it up, and stopped buying all these things.

He thought it was established that one could give too much calcium and not enough phosphorus, or the other way round, and thereby neglect the proper ratio. They must tell him if he was wrong, but he was quite sure that any animal which was fed on a normal diet, perhaps some dog biscuits or some meat, and a little milk, could never get rickets. He made this proviso, that it must not be kept in the dark; it must have sunlight. Of course, these Irish Setters were a large breed, and rickets seemed to him very largely confined to the large breeds; he supposed it was because they had got to make so much bone in the same amount of time, that if there was any lack of mineral in their diet then they could get rickets. Alsatians and big dogs of all sorts were much more prone. He had a case a month ago in a Labrador. She had three beautiful puppies, but by the time they were eight or nine weeks' old one of them began to roll about on his belly sprawling his hind legs out, unable to stand, and this fellow had been given all sorts of calcium and stuffs that the owner had bought from the chemist. He (Major Kirk) said, "Cut it out. Give the dog plenty of milk, raw meat and some biscuits," and—lo and behold!—in a fortnight the dog was standing up and running about, and the owner said it was marvellous.

They had got to draw some inference from that. He had seen cases in practice where he had given vitamin D and he had thought, "Well, that ought to put it right." But the rickets had gone on, and he just had not known why. He had found other cases where ultra-violet rays had been very effective. He exposed the hairless part of the abdomen to the mercury vapour lamp, starting with about two minutes only, seeing what the reaction was, and giving them as much as he could without causing dermatitis—sometimes up to ten minutes. His cases did improve with ultra-violet irradiation, but he did not give that alone. He usually used vitamin D as well; but he had to choose calcium and phosphorus preparations with great care in case he did not get the proper ratio.

Concerning cats, he had never seen rickets in cats at all. He did not know why they should not get it. There were plenty of old cats before the war that used to subsist entirely on twopennyworth of cats' meat thrown down the area on a skewer, and that was all that the cats had as a daily ration, and this was the only source of mineral they got—just a bit of meat on a skewer. Of course they knew there were plenty of dogs, too, which had nothing but a pure meat diet, and they did not get rickets. On the other hand, some dogs never had meat and they did not contract the disease either. He thought they tampered about with their food too much, and helped to produce the disease. Some people habitually gave young animals lots of liquid paraffin which in excessive amounts was also rachitogenic and for this reason. They knew there were many people who thought that if they gave a spoonful of liquid paraffin every day their animal was never going to be ill, but apparently the theory was that if one lined the intestine with so much non-absorbable oil it prevented the absorption of minerals from the food in the intestine. Whether that was true or not, he did not know, but he always warned clients, when they were giving paraffin every day of their lives, to desist. Oatmeal, also, appeared to be recognised as rachitogenic.

Those were a few practical recollections which occurred to him as he sat listening to the discussion. He had not really had much time in the last week to study anything. There had been such a multitude of papers that he had not been able to give the subject any consideration. Was there one who would like to contribute further to this discussion?

If not, he would ask Dr. Helgebostad kindly to reply.

The Reply

Dr. A. Helgebostad: He wished to express his best thanks to Professor Worden, Mr. Alan Wilson and Dr. Morris for their interesting remarks on the paper.

The dogs used in the experiments were German schäfers and Norwegian elf dogs of both sexes.

In the foxes fed Harrison and Mellanby's diet or the meat diet they found no difference in the histopathological picture. The diagnosis of rickets was based mainly on the pathological anatomical findings in the autopsied animals. They had not done any analyses of phosphatase in the dogs' blood.

To secure the animals against vitamin A deficiency they gave them carotene.

As to avoiding vitamin B deficiency, the experimental animals received a supply of dried brewers' yeast on Steenbock and Block's diet and of bakers' yeast on Harrison and Mellanby's diet.

He would like to point out, if one wanted to get rickets in one's experimental animals there must be a minimum of calcium or phosphorus in the diet. Harrison and Mellanby's diet contained only 0·4 per cent. Ca. If the animals got plenty of calcium and phosphorus they would not become rachitic in spite of the absence of vitamin D.

As far as they knew, there was agreement among the different investigators concerning the effect of vitamin D_2 and D_3. It was the same for both in mammalia, but in poultry vitamin D_3 was about 40 times as potent as D_2.

The Chairman: Thank you very much.

Professor Alastair Worden: If he might make a further remark; Dr. Helgebostad had not answered Mr. Wilson's question about whether he had any experience of using vitamin D_3 in the dog or the fox. He wondered whether their colleague from America would perhaps enlighten them on that point. He believed that the differences between large dogs and small dogs in their relative as well as total requirement of vitamin D, demonstrated by Morgan and other workers in the States, concerned vitamin D_2; and the suggestion had been made that there was a difference between vitamin D_2 metabolism in large and small dogs. So far as he was aware the same differences had not been demonstrated for D_3. From a practical prophylactic point of view, if in fact vitamin D_3 was almost as efficacious as D_2 for the dog, and if there was no difference between the large and small dogs in the utilisation of D_3, druggists and practitioners would probably be advised to change over to it. He did not know whether Dr. Morris had any information on that point. It was very important from a practical point of view because D_3 was not highly expensive as compared with D_2 in this country.

Dr. Morris (United States): D_3—he presumed that referred to the irradiated dehydro-cholesterol such as was manufactured in his country for use in poultry rations? Was that correct?

A vitamin D_3 plant happened to be in his home town, New Brunswick, N.J., and the biochemist in charge of the work was Dr. James Waddell, an alumnus of the University of Wisconsin. He had discussed this problem on a number of occasions with workers—that was, concerning the efficacy of vitamin D_3 and other forms of vitamin D and the effect on calcium and phosphorus absorption. They did not have specific information for the dog or for the fox. They did have it of course for poultry and other species. He could tell them this: there were a number of feed manufacturers in America buying vitamin D_3, and a large number of feed manufacturers were relying on the irradiated D in yeast as a source of vitamin D, and the incidence of clinical rickets appearing in practice was very small. Fifteen to 20 years ago they used to see quite a lot of rickets in dogs but clinically they rarely saw it any more. To see rickets in the dog it was necessary for them to produce it experimentally on a research farm.

Referring to the work of Morgan, a Californian, on the requirements of large breeds versus small breeds and their susceptibility to rickets, his criticism of Morgan's early work would be that the balance of the basal diet employed at the time that work was done, was based on biological knowledge of the requirements of the dog up to that point. Since that time, however, the nutritive requirements of the dog had been greatly elucidated, and he thought before any conclusions could be drawn now, it would be necessary to repeat that work. His personal opinion would be that one of the reasons rickets might appear more commonly in the large breeds was that people might stint on the food intake of the bitch at the time she was in whelp, also they might restrict the diet of the growing puppies, not realising the tremendous amount of available energy that was required by the young growing puppy of one of these larger breeds. They just did not give enough food to them with calcium and phosphorus in proper balance, and as a result they saw deformed bones in those breeds. In that connection they had noted some of the splay-footed condition, as they called it. This condition, common in the large breeds, had nothing to do with mineral metabolism or the feeding of vitamin D; it seemed to be a protein and fat utilisation problem and could be corrected. He did not know whether he had answered the question?

The Chairman thought they would agree with him that they had a very interesting, if abbreviated, discussion.

They were much indebted to their two Norwegian colleagues for imparting their knowledge to them, and to Dr. Helgebostad for coming so far to address them. It was his privilege, therefore, heartily to congratulate and sincerely thank the joint authors, the two openers and Dr. Morris for helping to make their meeting a success.

A SEMINAL DEFECT ASSOCIATED WITH STERILITY OF GUERNSEY BULLS

By

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The purpose of this communication is to record the identification of a seminal defect which we believe is indicative of the existence of a specific type of male sterility in the Guernsey breed. Working independently in the investigation of infertility of cattle, we have examined semen samples from a total of 463 bulls, including 89 of the above breed. The condition to be described was encountered in 12 of these which were all young bulls presented for examination with a history of total sterility.

The characteristic feature observed on microscopical examination of semen from the twelve animals was the virtual absence of intact spermatozoa, the ejaculates containing only free heads and tails. The general features presented on examination of stained smears are illustrated in the accompanying photo-micrographs. A total of 73 ejaculates was examined from the 12 bulls and 6 were studied by repeated sampling over periods of 1 to 8 months. The findings disclosed no variation from the typical pattern described. Observations were made on macroscopic appearance, initial motility, density and viability of whole semen; the morphological characters were studied by examination of fresh semen diluted in normal saline or Baker's solution and in stained smears.

Motility ratings on microscopical examination of whole semen gave low values, although a distinct wave motion was apparent in some samples and there was, in general, little indication of the grossly abnormal character of the ejaculates. Density values fell within the range 100,000 to 900,000 sperms per c.mm. and motility was maintained for periods varying from 36 to 116 hours' duration on storage of whole semen at 4° C.

Examination of diluted semen immediately after collection showed that the characteristic separation of the individual spermatozoa observed in stained smears was already evident and that the motility shown by the sample was due to the active movement of free tails, which on casual examination resembled intact spermatozoa, the "heads" being represented by a loop formation of the midpiece. More detailed observations of the morphological characters were made in studies of stained preparations. The proportion of intact spermatozoa never exceeded 5 per cent. of the total examined in any one sample. Among the separated elements the heads were of uniformly normal appearance, except for a small proportion showing the presence of a deeply staining body at the neck. All heads showed a deep indentation, marking the point of separation from the tail. In contrast to these findings the separated tails included a very high proportion of abnormal forms which, characteristically, showed a thickening of the anterior extremity of the midpiece associated with varying degrees of coiling of the midpiece and tail proper.

The identification of such an apparently specific type

J. L. HANCOCK and D. H. L. ROLLINSON : A Seminal Defect Associated with Sterility of Guernsey Bulls

IRON HAEMATOXYLIN × 2,500

IRON HAEMATOXYLIN × 800

of male infertility within a particular breed naturally suggested that it might be of genetical origin. Examination of pedigrees of the 12 bulls has failed to show any close linkage, the only ancestor common to all being seven generations removed in one case. As this degree of common ancestry might well exist in any series of bulls within the breed, there is no conclusive evidence to hand that the defect is an inherited one. It should be noted, however, that we have identified the condition, in two or more bulls, in each of four herds where there was evidence of some inbreeding in the direction of the common ancestor referred to. Further, we have been unable to identify any other common factor which might be of etiological significance. The possible influence of dietary and environmental factors receives little support from our observation that three of the sterile bulls were all bred in one herd, and that two of these had been sold within a month of birth and reared in widely different localities under different systems of management. Finally, it is of interest to note that Lägerlof (1934) has commented upon the association of a high proportion of abnormalities of the midpiece in spermatozoa from bulls suffering from an inherited testicular hypoplasia.

Although further work will be needed to establish the nature and origin of the condition, there appears to be ample evidence of the breed specificity of the condition and, it may be added, this conclusion finds some support from the observations of Wilson (1946) and Haq (1949), who have each recorded identification of the condition in individual Guernsey bulls, and those of Makinson (1949), who has recently identified the condition in two bulls of this breed.

Acknowledgment.—We wish to thank Dr. D. J. Trevan, Wellcome Physiological Research Laboratories, for the photomicrographs.

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THE "BOTTLE MENTALITY"

Closely bound up with the prevalence of incapacitating sickness was the vogue for "patent" medicine, said Dr. A. G. Mearns, senior lecturer in hygiene in the University of Glasgow and medical adviser to the Scottish Council for Health Education, in his address to the annual conference of the Royal Sanitary Association of Scotland. That the average proprietary medicine was innocuous was not questioned, he continued. The criticism to be levelled against them was that they encouraged in the less discerning members of the public a completely wrong attitude to the treatment of disease. A "bottle mentality" was created, as a result of which, for example, grave intestinal maladies in their early stages were treated by proprietary "indigestion" mixtures, and incipient tuberculosis by cough syrups. While it was in the public interest that there should be on the market a suitable range of simple remedies for use in the appropriate conditions, it was highly desirable that these should be standardised. The greatest need was to convince the public that good health came from a correct regimen, and that ill-health demanded medical care and not self-treatment.—*Pharm. J.*

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Weekly Wisdom

The old learning, the love of a craft for its own sake, of truth not dressed for the party, of service with individualism, is hurrying away from the earth and soon will be only a footprint,—R. C. Robertson-Glasgow: "46 Not out."

CLINICAL COMMUNICATIONS *

C. W. CARTWRIGHT, M.R.C.V.S.,

Dyserth, Flints.

I.—FOREIGN BODY IN A MARE

The subject was a very useful eight-year-old Vanner mare to which I was first called on June 29th, when the chief symptoms shown were dullness, inappetence, elevated temperature (104°) and slightly irregular heart sounds.

Sulphonamides and cardiac tonics were administered to which she responded, but later her membranes became distinctly icteric. Cholagogues were given. She became normal and was turned out. On July 18th she became dull again with icteric membranes and a slight temperature. Again she responded to treatment and later was put to light work. On August 3rd dull pain was noticed for the first time, but when seen the next day she was normal and when turned out she was fresher than usual.

In view of the recurring attacks and as she was insured, it was arranged by the Insurance Company that Mr. Llewelyn Jones, of Denbigh, should see her, but we agreed to defer the consultation until acute symptoms again were manifested.

The same night she showed acute abdominal pain for the first time. Sedatives were administered. Next day we saw her in consultation. Her condition was now critical; irregular weak pulse, temperature elevated (103°) and constant acute pain. Rectal examination disclosed an enlarged ovary, but it was agreed that the lesion was in the small bowel, possibly a calculus or growth. Sedatives were administered, but she died the next morning.

Owing to a misunderstanding the *post-mortem* examination was not satisfactory, most of the organs having been removed and disposed of. However, the causal lesion luckily was found and was in the form of a small mass of organised tissue measuring about 4 inches by 3 inches lying between and adherent to two loops of the ileum. When incised it was found to contain a small quantity of pus and a thin piece of wire netting about 3 inches long. Unfortunately the liver was not available, but the slight jaundice was probably due to a catarrhal condition of the duodenum extending to the bile duct.

This is my first experience of wire in a horse, but one wonders how many indefinite colic cases may be due to a similar cause.

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II.—BLACK QUARTER

The subject was a yearling Shorthorn heifer in good condition which showed clinical symptoms of black quarter. There was a large swelling with crepitant areas over the left hindquarter causing marked lameness and systemic disturbance. Black quarter had occurred on the farm.

One million units of penicillin were injected into the swelling and repeated daily in decreasing doses until by the end of the week she had received three million units. A formalin mixture also was prescribed.

By this time she was decidedly better, the swelling had

(*concluded at foot of next page*)

* Made to the "Clinical Forum" meeting of the North Wales Division, N.V.M.A., at Llangollen, June 30th, 1949.

ABSTRACTS

[The Prolonged Administration of Thyroxine to Cows, with particular reference to the Effects on Thyroid Function and on Pituitary Thyrotrophic Hormone. McQUILLAN, M. T., TRIKOJUS, V. M., CAMPBELL, A. D, & TURNER, A. W. (1948.) *Brit. J. Exp. Path.* **29.** 98.]

Fifteen non-pregnant, Shorthorn cows, aged about 2½ years in good store condition, which either had aborted or, following immunisation with living *Brucella abortus* "Strain 19" vaccine, resisted exposure to virulent *Br. abortus*, were used in the experiments. The experiments were carried out from mid-March to mid-July, 1946, when the animals had excellent pasturage. Twelve of the animals were matched according to weight and one of each pair allocated to treated and control groups, the three larger unmatched animals serving as a sub-group of treated animals. The experiment covered four periods, the first 16 days being a training period in order to accustom the animals to the routine of handling and clinical examination. In Period 2 (15 days) the animals were injected subcutaneously three times weekly with 15 mg. of thyroxine dissolved in physiological saline, in Period 3 (25 days) 20 mg. thyroxine three times weekly and in Period 4 (30 days) 40 mg. thyroxine three times weekly. Each animal received during the 70 days of treatment 805 mg. of thyroxine, equivalent to 11.5 mg. mean daily intake. Observations and treatment were carried out at the same time each morning. Respiratory rate, heart rate and rectal temperature were recorded daily, the animals were weighed weekly, and every second week 5 ml. of blood was drawn into heparin for estimation of specific gravity by the copper sulphate method and for agglutination tests against *Br. abortus*. The animals were destroyed four days after the last injection. One thyroid from each cow was assayed for thyroxine. The anterior pituitary lobes from both groups were assayed for thyrotrophic potency in two-day-old White Leghorn cockerels. All results were examined statistically by means of the analysis of variance.

About three weeks from the commencement of thyroxine administration, the treated animals became excitable and restless. They appeared unthrifty, and although they did show weight gain throughout the experiment, the weight increase was only 30 per cent. of that gained by the controls. The maximum mean respiratory frequency (77/min.) was greater in the treated group than in the control group (57/min.). The maximum mean cardiac frequency (97/min.) in the treated animals was also greater than in the control group (76/min.). These relatively high values probably may be associated with the fact that the animals were brought in from the field for examination in a crush. During the course of the experiment, the udders of the treated animals developed to a state resembling 6-7 months' pregnancy, the teats became turgid and up to 30 ml. of milky secretion could be expressed. The specific gravity of the blood of the control group increased during the experiment, while in the treated animals it did not. There was no significant change in agglutination titres of sera against *Br. abortus* either during the course of the experiment or as between either group.

Microscopical examination of the thyroid glands showed a significant lowering of the acinar cell height in the treated animals and biochemical estimation a significant lowering of the ratio of thyroxine to total iodine. This depression of thyroid function in the thyroxine-treated animals was confirmed by chick-thyroid assays. These showed an absence of thyrotrophic hormone in the anterior pituitary of the treated animals in contrast to its presence in the control animals. The anterior pituitary lobes of the treated animals showed no thyroid inhibitory factor.

Other organs showed normal appearance at *post-mortem* examination and, later, on histological examination, except in the mammary gland of the treated animals where histological examination showed little adipose tissue, but extensive proliferation of ducts without true alveolar development. The vertebral columns of the treated animals were extremely hard on "sawing down" and were compared by the butchers to "old cows" of 7-8 years of age. This latter finding is to receive further special investigation.

F. R. B.

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[Congenital Hydrops in Baby Rabbits: a Hereditary Foetal Erythroblastosis. (Trans title) NACHTSHEIM, H, & KLEIN, H. (1947.) *Abdh. dtsch. Akad. Wiss. Berl.* No. 5.]

This paper gives a detailed account of finding, in a group of rabbits, as a result of inbreeding experiments, new-born young showing generalised oedema and erythroblastosis, and in some cases erythroblastosis without oedema. Both conditions were always fatal. The paper is divided into two parts. The first is a description of the inheritance of the condition, the second gives a full account of the naked-eye appearance of the affected young and of the histology of each tissue.

The factors underlying the inheritance are stated to be not yet fully elucidated. All the affected young derived from one albino buck which was inbred with its daughters and granddaughters. Hydrops in the young was formed from the second generation of inbreeding. Affected young were found in the same litter as normal young, and made up about 3 per cent. to 23 per cent. of the total progeny of a doe. At first it seemed likely that a recessive "hydrops" gene was responsible, but the numbers of the affected young were very much less than would have been expected if this had been the only factor controlling the inheritance. The mother and her genotype seemed to be of greater importance than the father's genotype and an additional conditional factor is suggested.

Rabbits with hydrops and erythroblastosis or with erythroblastosis without hydrops, were almost always born alive, but always died at or within a few hours of birth. They were all albino. The oedema was generalised and affected all tissues capable of oedema. There was a marked degree of erythroblastosis, shown principally by extra-medullary haemopoiesis, much of which was in the liver. The spleen was almost always enlarged and again showed much erythropoiesis. Nucleated cells of the

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become localised and later showed signs of pointing. Eventually it was opened, discharging thin, sanguinous pus, and it was evident that in due course the whole mass would slough.

In rather less than a month from the onset of the illness, the mass of muscle, comprising the semitendinosus and semimembranosus, was removed intact through the enlarged abscess wound.

As may be expected, a large local depression resulted, but it gradually filled up and the animal made a good recovery.

I cannot specify the causal organisms, but it is my first experience of successful penicillin treatment in a clinical case of black quarter.

erythroid series were the predominant features of the peripheral blood picture. Stored iron could be demonstrated regularly and was held to point to increased blood destruction. Jaundice was not seen.

A very close parallel is drawn between the findings in the hydropic rabbits and those in haemolytic disease of the new-born in humans due to isoimmunisation of pregnancy by red cell antigens of the Rh. series. It is suggested that this hydrops in rabbits has a similar aetiology, and that a gene responsible for abnormal permeability of the placenta is a conditional factor which must also be present before foetal red cell antigens can reach the mother's blood stream.

A report of preliminary serological investigations by Dahr and Knüppel is appended. Tests on the rabbit stock using Rh. agglutinins were negative. Guinea-pig, anti-rabbit, red cell antisera were prepared, using the blood of the buck with most hydropic young. When the resulting antiserum was absorbed with red cells from a doe bearing hydropic young a grouping serum was obtained which agglutinated the red cells of all the bucks of the hydrops stock. The red cells of most of the does bearing hydropic young were negative. A few were positive, but this is explained by suggesting for the rabbit a series of multiple alleles governing their red cell antigens.

D. H.

REVIEW

THE P.J. POISONS GUIDE

A Guide to the Pharmacy and Poison Rules and the Pharmacy and Medicines Act, 1944, with an Extended Poisons List

In this publication of 50 pages we have a concentrated essence of the 1933 Act with the various additions up to date. It is written specially for the pharmacist in order to enable him to discharge his duties to the community faithfully and well in the shop or hospital dispensary.

Consequently, we veterinarians find in it no special reference to our obligations as far as our work is concerned. Your reviewer, who writes also as a pharmacist who has acquired some knowledge of the Act, realises the complexity of the subject, and therefore has a fair idea of the responsibility that rests on the pharmacist as the intermediary between those who are concerned in the realm of poisons—the veterinarian, the doctor, the dentist, and the general public. To the veterinary surgeon the valuable part of the publication is the Extended Poisons List, which shows at a glance whether the poison is an ordinary one which does not demand anything extra, or one under Schedule 1 or Schedule 4, or a dangerous drug.

The Secretary of State for Home Affairs is the ultimate authority as to what substances should be on the Poisons List. But the Home Secretary—in all likelihood a layman in these matters—can act only after consultation with or on the recommendation of the Poisons Board. This Board is made up of about 16 members who represent various interests: the Home Secretary, the Secretary for Scotland, Ministry of Agriculture and Fisheries, Ministry of Health, Government Chemists, Institute of Chemistry, College of Physicians of London and Edinburgh, General Medical Council, British Medical Association, and the Pharmaceutical Society.

The provisions of the Act relating to poisons may be modified by the Home Secretary after consultation with or on the recommendation of the Board.

These are terms in constant use in the Act which are fully explained, but in this Guide a knowledge of their meaning may be more or less taken for granted. To give a few examples—poison, dispensing, compounding, authorised seller of poison, listed sellers authorised persons.

Poison means, in law, any substance in the Poisons List.

Dispensing means the supply of medicine as poison in accordance with a prescription of a doctor, veterinary surgeon or dentist.

Compounding—the mixing of drugs or poisons, but not in accordance with a prescription as above.

The Medical & Pharmacy Act, 1941, states, in connection with Drug Stores, that drugs must not be compounded or dispensed on the premises.

Authorised Seller of Poisons—Applies to private pharmacist, a separate body, a representative of a deceased pharmacist.

To become an authorised seller a pharmacist must have his premises registered with the Pharmaceutical Society and his Certificate must be exhibited. A corporate body may become an authorised seller by appointing a pharmacist in charge. In the case of a deceased pharmacist a representative may be appointed, *e.g.*, a solicitor, accountant, or executor. He will be responsible in law and must appoint a registered pharmacist to be in charge.

Poisons are divided broadly into two main classes, Part 1 and Part 2. For the most part Part 1 poisons are used in medicine and the authorised seller only can deal in them. Doctors, veterinary surgeons, and dentists have a right to prescribe and dispense them but not to sell. If a veterinary surgeon sells, say, aconite powder he breaks the law. Part 2 poisons, such as the acids above 9 per cent., sheep dip caustics, can be sold by a listed seller as well as by an authorised seller. The former gets his licence to sell from the local Council. Both Part 1 and Part 2 poisons are in the main divided into first Schedule and not first Schedule. A few are Schedule 1 only—such as ergot, strophanthus and nux vomica, provided it contains more than 2 per cent. strychnine.

In the case of S.1 poisons there are special restrictions in connection with their sale. The authorised seller must know the customer. He must be satisfied that the purchaser is a person to whom a poison can be sold. Before an S.1 poison can be handed over, certain particulars must be given, such as name and address, date, name and quantity of poison, purpose for which it is stated to be used, and the purchaser must append his signature to the Poisons Register. When a poison is required for trade, business or professional purposes, a signed order giving certain particulars will suffice. This is pinned to the Register in place of a signature. This is the procedure to be followed by a veterinary surgeon when he requires an S.1 substance for his professional work, but if he wants an arsenical weed killer he is a member of the general public and the ordinary routine has to be gone through. If his gardener requires the weed killer a signed order will do because killing weeds is a part of his business. All poisons must be in containers that are impervious and must be able to withstand ordinary risk of handling.

In all cases the labelling provisions must be adhered to:—Name of Poison, Name and address of seller, word "Poison" in red if S.1. The words "Not to be taken —For external use only" have their own special significance, and these words must be in frames so that the lay person will notice them.

There are also rules regarding storage, transport, and colouring. The rule *re* storage does not apply to the surgery of a veterinary surgeon, but it would be well if the veterinary surgeon imposed this rule upon himself.

Dispensed Medicine.—The veterinary surgeon when dispensing animal medicines which are Schedule 1 must keep a record and when writing a prescription for the pharmacist to dispense must observe certain rules:—He must state the name of the person who is in charge of the animal, the name and quantity of poison and use the words to be put on the label: "For animal treatment only." Reference to the Extended Poisons List will indicate whether it is S.1, S.4 or D.D.A.

Schedule 4 Drugs.—Barbiturates, sulphonal, sulphonamides, etc., can filter to the general public only via a prescription written by a veterinary surgeon, doctor, or dentist, but if required for trade purposes the pharmacist can supply as an S.1 poison by complying with Register Regulations, a signed order sufficing.

The Guide furnishes concise particulars and gives the official view as to when a person is carrying on a trade or business. A farmer who keeps sufficient fowls to yield a supply of eggs for the household cannot get an S.4 drug in this way. But if he keeps a number that will enable him to sell eggs for profit—that would be regarded as trading. In writing prescriptions for S.4 drugs the veterinary surgeon must adhere rigidly to the rules given in the Guide. If, for example, he does not date or initial it, the pharmacist will be breaking the law in dispensing it. The pharmacist must also copy the prescription, stamp it, and date it, in such a way that the stamp cannot be removed. If the word "repeat" appears in the prescription, there must be no more than two supplies; when dispensed for the last time, the prescription is retained by the pharmacist and kept for at least two years.

Dangerous Drugs.—A veterinary surgeon is authorised to be in possession of these drugs.

Raw opium, coca leaf, and Indian hemp must be used only for making preparations for veterinary purposes. They cannot be prescribed. It is medicinal opium, tincture of opium, ext. Indian hemp, etc., that he prescribes or dispenses.

Registered veterinary surgeons may issue prescriptions, give orders and may be supplied with dangerous drugs under the same conditions as doctors. They must endorse prescriptions "For animal treatment only," and in place of the patient's name and address give those of the person to whom the article is to be delivered, *i.e.*, to the person in charge of the animal.

A prescription written by a veterinary surgeon can be dispensed by the pharmacist without question, bearing in mind the following rules as to what must be recorded:—

(*a*) Name and address of the person in charge of animal.
(*b*) Date in veterinary surgeon's own writing.
(*c*) Total amount of drug to be supplied or the amount of preparation of the drug as contained in a B.P. or B.P.C. preparation.
(*d*) Usual signature—surname in full.
(*e*) Veterinary surgeon's postal address.
(*f*) If repeats are ordered, the intervals between each supply; there must not be more than three supplies altogether.

This prescription is not returned to the owner. It differs in this respect from S.4 prescription.

From what has been said it is evident that, from all points of view, this booklet should be in the hands of every veterinary practitioner.

QUESTIONS IN PARLIAMENT

Attested Cattle (Scheme)

Mr. Hurd (October 27th) asked the Minister of Agriculture if he has now agreed with the representatives of farmers and the veterinary profession on a scheme for the establishment of clean areas where only attested cattle will be permitted; and when a start will be made.

The Minister of Agriculture (Mr. Thomas Williams): I am afraid that I am not yet able to add anything to the reply given to the hon. Member on April 4th.

Mr. Hurd: Can the Minister give us some reason for the prolonged delay in starting this scheme for the eradication of tuberculosis?

Mr. Williams: As the hon. Member is aware, discussions have been proceeding for quite a long period, and the question of compensation and finance generally, perhaps may have caused the delay.

Major Sir Thomas Dugdale: Is it proposed to include the postponing of this scheme under the £6 million cuts announced earlier this week?

Mr. Williams: It is quite conceivable that there may be some deferment for a short period.

Sir T. Dugdale: Would not the Minister agree that it is a very undesirable way of saving expenditure when the whole object is to increase better class milk to the country?

Mr. Williams: I do, of course, but all economies are more or less painful.

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Horse Slaughter (Committee's Report)

Mr. Gammans (October 24th) asked the Minister of Agriculture when the committee which he has set up to look into the export of horses and their transportation and slaughter in this country is likely to report.

Mr. G. Brown: The chairman hopes to have the report ready early next year.

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Dangerous Dogs

Mr. Warbey (October 20th) asked the Secretary of State for the Home Department whether, in view of frequent attacks by greyhounds on other animals in public places, he will introduce amending legislation to enable local authorities to make by-laws for the muzzling and control of greyhounds.

Mr. Ede: On the information before me I have not sufficient evidence to show that the existing law relating to dogs which are dangerous and not kept under proper control is inadequate.

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Docking and Nicking of Horses Bill

In the House of Lords on Wednesday of last week the Docking and Nicking of Horses Bill was read the third time and passed.

NOTES AND NEWS

Diary of Events

Nov. 7th.—R.C.V.S. Pharmacology Examination (oral and practical), London and Dublin.

Nov. 7th.—Meeting of the Farmers' Club, Royal Empire Society, 2.30 p.m.

Nov. 9th.—Meeting of the N.V.M.A. Technical Development Committee, 11 a.m.

Nov. 9th.—Meeting of the Editorial Executive Committee, 2.30 p.m.

Nov. 11th.—R.V.C. Students' Union Society Commemoration Ball. (See Notice.)

Nov. 12th.—Dinner of the Southern Counties Division, N.V.M.A., at Southampton (Polygon Hotel), 7.30 p.m.

Nov. 12th.—Meeting of the Biochemical Society, at the London School of Tropical Medicine, Keppel Street, W.C.1, 11 a.m.

Nov. 16th.—Meeting of the Royal Microscopical Society, in the Hastings Hall of B.M.A. House, Tavistock Square, W.C.1, 5.30 p.m.

Nov. 18th.—Annual Meeting of the North Wales Division, N.V.M.A., at Ruthin (Wynnstay Hotel), 2 p.m.

Nov. 23rd.—Dance of the Ayrshire Division of the Victoria Veterinary Benevolent Fund Ladies' Guild, at Western House, Ayr.

N.V.M.A. Committee and Council Meetings

The following are the arrangements for meetings of Committees and Council of the Association to be held in London in November:—

Tuesday, November 15th:

11 a.m.—Parliamentary and Public Relations Committee.

2 p.m.—Organising Committee.

4.15 p.m.—Home Appointments Committee.

Wednesday, November 16th:

10 a.m.—Veterinary State Medicine Committee.

2 p.m.—General Purposes and Finance Committee.

Thursday, November 17th:

11 a.m.—**Meeting of Council.**

Friday, November 18th:

10.30 a.m.—**Special Meeting of Council.**

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PERSONAL

Appointment.—Mr. Walter Plowright, M.R.C.V.S., who graduated at the Royal Veterinary College in 1944 and since February, 1948, has been demonstrator in pathology there, has been appointed to the Colonial Service as a Veterinary Officer (Pathology) in Kenya.

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Births.—Frost.—On October 22nd, 1949, to Marguerite, wife of H. Frost, Fairmile, Oathall Road, Haywards Heath, Sussex, a son, Richard Martin.

Boundy.—On October 26th, 1949, to Catherine, wife of Terry Boundy, B.V.Sc., M.R.C.V.S., of Crag Bren, Montgomery, N. Wales, a sister for Christopher.

McTaggart.—On October 25th, 1949, at Leeds, to Iselle (*née* Jackson), wife of H. Scott McTaggart, B.Sc., M.R.C.V.S., The Hollies, Appleby, a daughter—Judith Anne.

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Marriages.—Arnold—Sharp.—On July 2nd, 1949, at the Garrison Church, Up-Park Camp, Kingston, Jamaica, Roger, second son of Mr. and Mrs. G. T. Arnold, of Sittingbourne, Kent, to Prudence, second daughter of Mr. and the late Mrs. T. H. Sharp, of Christiana. Present address, Veterinary Investigation Dept., Dept. Agriculture, Hope, Kingston, Jamaica, B.W.I.

McKenzie—MacLean.—On October 5th, 1949, at West Parish Church, Inverness, Robert L. McKenzie, M.R.C.V.S., of Inverness, to Elizabeth D. MacLean, of Inverness.

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Dr. J. C. Broom's Milroy Lecture on "Leptospirosis."—At the quarterly comitia of the Royal College of Physicians, Dr. J. C. Broom, Wellcome research bacteriologist, was appointed Milroy lecturer for 1951, his subject being "Leptospirosis." We congratulate Dr. Broom on this distinction. He was part author, with Miss Joshua, of the paper on this subject presented to Congress, N.V.M.A., published in our issue of October 29th.

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R.C.V.S. OBITUARY

Hodgson, John Fletcher, M.R.C.V.S., West Croft, Brigham, Cockermouth, Cumberland. Graduated Edinburgh, December 14th, 1926. Died October 25th, 1949.

Murnane, Richard J., Newcastle West, Co. Limerick, Eire. Graduated N. Edinburgh, March 30th, 1896. Died October 24th, 1949.

O'Gorman, Charles Lennon, Lt.-Col. A.A.V.C., Hargreaves Road, Manby, Queensland, Australia. Graduated Liverpool, December 20th, 1909. Died 1949.

Lt.-Col. C. L. O'Gorman, M.R.C.V.S.

The August issue of the *Australian Veterinary Journal*, just received, reports the "recent" death (date unspecified) of Lt.-Colonel C. L. O'Gorman M.R.C.V.S., who was a student at the University of Liverpool Veterinary School, graduating in 1909. After a period in general practice he accepted a position as Government Veterinary Surgeon in Queensland, and in April, 1914, was appointed to the Department of Agriculture in New South Wales. Mr. O'Gorman (the *A.V.J.* continues) was one of the first veterinary officers to leave Australia, and served in Egypt, Gallipoli, France and Belgium. He had a wide experience with units and in command of a Mobile Veterinary Section. On his return to Australia he resumed duty as a Senior Veterinary Officer in the Department of Agriculture, New South Wales, and was later appointed Chairman of the Board of Tick Control, carrying through extensive operations. "He was particularly gifted in his handling of men, and his staff held him in high regard. Professionally he was not only a sound diagnostician, but a capable administrator."

Following his retirement in 1944, Colonel O'Gorman took up farming in South-East Queensland.

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The Late Mr. William Crawford, M.R.C.V.S.

An Appreciation

Mr. Arthur H. Watson, of Bradford, writes of Mr. William Crawford, of Leeds, whose death we recorded with deep regret in our issue of October 22nd: "Will Crawford was a sound and very energetic practitioner, always inspiring confidence alike amongst his large clientele and his wide circle of friends. To all these his death comes as a tragic loss—as, indeed, in its nature also it was tragic, for he collapsed while attending a case and died within a few minutes.

"William Crawford was educated at Beith and qualified from Glasgow College in 1898. After posts as assistant in Glasgow and Birmingham he purchased the old-established practice of Mr. Robertson, of Leeds, in 1902. He became a member of the Yorkshire V.M.A. in 1902 and from that date until his death he was a regular attender, holding office from 1905-49, including the posts of President, Member of Council and Auditor. Also since 1939 he performed most valuable work as Petrol Liaison Officer. Our deepest sympathies go out to his wife (*née* Miss Annie Mitchell, whom he married in 1903), and others bereaved—his son, daughter-in-law and grandson."

Mr. Crawford's remains were cremated on October 15th at Lawnswood Cemetery, Leeds. The following were present at the cremation: members of the family, several clients and personal friends, while the Yorkshire Veterinary Medical Society were represented by Miss Woods (President), Messrs. J. Dixon, R. Dixon, R. Hayes, H. M. Holland, F. Lofthouse, A. McCarmick, J. W. Procter, F. C. Scott, P. L. Shanks, K. G. Towers and A. H. Watson.

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Mr. J. Fletcher Hodgson, M.R.C.V.S.

A Tribute

Friends and colleagues of John Fletcher Hodgson were shocked to hear that he died at his home at Brigham on the morning of October 25th, 1949.

Fletcher Hodgson was a Cumbrian by birth and upbringing and a graduate of the Royal (Dick) Veterinary College, Edinburgh, where he was held in the most affectionate regard by his fellows for his enthusiasm in athletic and social activities. He qualified in 1926 and succeeded his father in the practice at Brigham. A shrewd observer, quiet and unassuming, he was much thought of by farmers in a wide district. For many years he was a member of the Dumfries and Galloway Division of the National Veterinary Medical Association and was a frequent attender of meetings.

He is survived by his wife and two young sons, and to them goes the sincere sympathy of his colleagues. Among the great number of mourners at the service at Brigham Church on Friday, October 28th, were these members of the profession: Mr. J. Bolton, Gosforth; Mr. D. W. Macdonald, Brigham; Mr. Mackay, Cockermouth; Mr. W. G. Robinson, Carlisle; Mr. C. McNulty, Carlisle; Mr. J. E. Syme, Egremont; Mr. F. Lofthouse, Otley, and Mr. John Reid, Ministry of Agriculture and Fisheries, Carlisle.

J. R.

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PROPOSED FACULTY OF VETERINARY MEDICINE AT QUEEN'S UNIVERSITY

Minister Rejects Suggestion as "Not Opportune"

At Stormont, on October 18th, in reply to a question by Mr. McCoy, the Minister of Agriculture (the Rev. Robert Moore) stated that he did not consider the present an opportune time to suggest that a faculty of veterinary medicine should be established at Queen's University.

There were approximately 100 veterinary officers in the employment of the Ministry and roughly 70 veterinary surgeons in private practice in Northern Ireland. All received their qualifications either at Veterinary Colleges in Great Britain or at the Veterinary College in Dublin.

Since annual replacements for the Ministry and wastage in private practice were so small he did not consider the present an

opportune time to suggest that a Faculty be set up in Queen's University for the purpose of conferring degrees in veterinary medicine. The cost of setting up and running such a Faculty would far outweigh the need for it.

Moreover, even if a Faculty were set up there would be no guarantee that graduates would join the Ministry's staff or set up in private practice in Northern Ireland.

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UNIVERSITY OF EDINBURGH DEGREE IN VETERINARY SCIENCE

At the Graduation Ceremonial of the University of Edinburgh held recently, the following students of the Royal (Dick) Veterinary College graduated B.Sc. in Veterinary Science: Bowman, John George Wood; Finlay, Robert Andrew; Horseman, Matthew, and Malone, Gerald Samuel Robert.

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UNIVERSITY VETERINARY SCHOOLS

In the course of his address at the half-yearly meeting of the General Council of the University of Glasgow, the Principal, Sir Hector Hetherington, said that this year the university would spend £500,000 on extensions and new buildings. The incorporation of the Veterinary College had been effected with great harmony on both sides. Physically, the incorporation affected only first-year students, the others remaining at Buccleuch Street, but plans were being developed for new buildings at university sites in Glasgow and also in Ayrshire in collaboration with the West of Scotland College of Agriculture, and Sir Hector had no doubt that in a few years they would be able to point to a fine range of facilities for teaching and research.

The Bristol School

The writer of "University Notes" in the *Western Daily Press and Bristol Mirror* observes that the new School of Veterinary Science on the Coliseum site has a finished appearance to the casual glance, but much still remains to be done inside. "Laboratory equipment and fittings will be going in there very soon under the guidance of Professor C. W. Ottaway, who has been appointed Professor of Veterinary Anatomy. He has already arrived in Bristol from Cambridge University in order to supervise the later stages of the work. Another appointment for the new school is that of Mr. A. Carlyle, who comes from Sheffield University, and will be senior lecturer in veterinary physiology.

"The intention is to have the building completely ready by the beginning of the next session in eleven months' time. This session 25 veterinary students were enrolled, but their first year will be spent on elementary work in the Faculty of Medicine. Then will follow two years in the Coliseum building, and their fourth year will be spent at the University estate at Langford, where the practical work will be done.

"Although Langford will not therefore be invaded by students until 1952, it is already the scene of important research work. The Ministry of Agriculture Veterinary Investigation Centre has moved there from Bracken Hill, Leigh Woods, as tenants of the University, and probably will have permanent laboratories there."

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THE ANIMAL HEALTH TRUST

Forthcoming Meetings in Kent

Mr. J. W. Bruford, M.R.C.V.S., President of the N.V.M.A., will be one of the speakers at a public meeting, arranged by the Kent County Committee of the Animal Health Trust, to be held in the Cornwall Hall, Sevenoaks, on November 21st at 7 p.m. The speaker at a meeting to be held in the County Hotel Hall, Canterbury, on November 30th, at 7 p.m., will be Dr. W. R. Wooldridge, M.Sc., M.R.C.V.S., Scientific Director of the Trust. At both meetings the films, "Sterility in Dairy Cattle" (winner of a first prize in the International Film Festival at Venice) and "Hygiene on the Farm" will be shown. A discussion will be held and questions invited after the showing of the films.

Surrey Executive Committee

Plans for a country-wide campaign to publicise the work of the Trust, to recruit members and to raise funds for development of the work were discussed at Guildford recently by the Surrey Executive Committee recently formed to support the Trust.

Messrs. E. P. Barrett and J. Chapman were appointed vice-chairmen of the committee and it was decided to ask the manager of Lloyds Bank, Guildford, to become hon. treasurer. Rules were adopted.

The committee unanimously decided to recommend Lieut.-Col. A. C. W. Upton, of June Cottage, Merrow Woods, Guildford, to headquarters for appointment as paid organiser for Surrey, with possibly additional territory later.

The committee appointed sub-committees, with power to co-opt, for the following sections: equine; canine; poultry; pigs; fur and feather; livestock.

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VISIT OF ITALIAN VETERINARIANS AND AGRICULTURISTS

A group of seven Italian veterinarians and agriculturists recently spent a ten days' visit in this country under the auspices of the British Council. During their stay they studied British methods of treating and preventing infective diseases in animals, and visited research institutes. On Saturday, October 22nd, they visited the Milk Marketing Board's Artificial Insemination Centre at Whiligh, Sussex, and on Monday, October 24th, the National Institute for Research in Dairying at Shinfield, near Reading, in the morning, and the Ministry of Agriculture Cattle Breeding Centre in the afternoon.

The whole of Tuesday, October 25th, was passed at the Dairy Show at Olympia. The five veterinarians in the party visited the Ministry of Agriculture Veterinary Laboratory at New Haw, Weybridge, on Wednesday, October 26th, and on the following day the whole group were at the Agricultural Research Council Field Station at Compton, Berks. On the 28th came a visit to Cambridge, which included the Department of Animal Pathology, and on the Saturday the party went on to the Equine Research Station, Newmarket, returning to London the following day. Their visit ended on Monday, October 31.

The party comprised:—Dr. Carlo Roetti and Dr. Elios Luisi, Provincial Veterinarians attached to the High Commission for Public Health and Hygiene; Count Alessandro Cavazza, a leading Italian cattle and horse breeder, Rome; Signor Ciarrocca, stock-breeder, Rome; Dr. Vincenzo Sanfile, head of the Artificial Insemination Department at Rome Zoo-prophylactic Centre; Dr. Bruno Baldelli and Dr. Gionisio Mura, assistants at the Zoo-prophylactic centres at Perugia and Sassaria.

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FORTHCOMING CONFERENCES ON PIGS

Under the auspices of The British Oil and Cake Mills, Ltd., a series of special national pig conferences has been arranged, at which Dr. Laurence M. Winters, of Minnesota, will give addresses—illustrated by coloured films—on "The Modern Use of Tools—Inbreeding, Crossbreeding and Selection for Pig Improvement."

Dr. Winters, who was selected by the Morrison Award Committee of the American Society of Animal Production as the 1948 recipient of the Morrison Award, is a leading research worker in the field of animal breeding. Since 1928 he has been a member of the animal husbandry staff of the University of Minnesota, where he is now Professor in charge of Animal Breeding. Outstanding among the many research projects he has directed are the studies in crossing breeds of swine, studies in the physiology of reproduction and prenatal development of farm animals, studies in techniques in artificial insemination of cattle and studies in methods of breeding for the improvement of farm animals.

The first conference will be held in the Pollok Hall, Edinburgh, on Wednesday, November 30th, at 7.30 p.m., under the chairmanship of Professor J. Russell Greig, PH.D., M.R.C.V.S., F.R.S.E., with Dr. A. D. Buchanan Smith as the opener of the general discussion. The venue of the second conference is the University, Bristol, where, at 2 p.m. on Thursday, December 8th, the chair will be taken by Dr. H. I. Moore, M.Sc., PH.D., N.D.A., DIP. AGRIC., with Mr. W. T. Price, M.C., B.Sc., P.A.S.I., N.D.A., N.D.D., and Dr. J. E. Nichols, PH.D., M.Sc., F.R.S.E., opening the discussion. The final conference takes place in the Small Guildhall, Cambridge, on Wednesday, December 14th, at 2 p.m. The Chairman will be Professor W. C. Miller, M.R.C.V.S., F.R.S.E., and Dr. Winters will be followed by Dr. J. Hammond, C.B.E., D.Sc., M.A., F.R.S., and Mr. W. A. Stewart, O.B.E., M.A., B.Sc. (AGRIC.).

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A CHILLINGHAM HEIFER

Fortunately for the herd of wild white cattle which for many centuries has roamed the area around Chillingham, the Northumberland home of Lord Tankerville, a heifer calf has at last been born, after a long run of bull calves.

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Last winter a Danish scientist put cows on a diet in which seaweed was substituted for cattle cake. He found that the butter yield was of a higher quality than that of cows fed in the normal way.—*Farmer and Stock-breeder.*

LIVESTOCK EXPORT GROUP

The following items of interest to our readers appear in the Report for the quarter ended September, 1949:—

Argentine Trade Agreement

As the result of discussions between the Chairman and the Secretary for Overseas Trade, the Executive was asked to express its opinion on the allocation made in the Argentine Trade Agreement for the annual importation of "live animals" to the value of £250,000.

This sum was considered to be insufficient in view of the backlog of animals under obligation since the latter half of 1948, purchases at and since this year's Perth Sales, and also the large number of bloodstock purchased.

Members were of opinion that if the value of these outstanding purchases were to be deducted from the allocation, the balance available for purchases during the rest of the period, as laid down in the Agreement, would, in all probability, be insufficient. Bloodstock sales in the latter half of this year and particularly the Perth Sales in early 1950 might be seriously jeopardised in the absence of information as to whether requests for the grant of import licences would be met. Difficulties were also foreseen in obtaining payment by those exporters who sent livestock to Argentina for auction there should animals be sold at a price higher than that indicated in the import licences.

The matter was submitted in detail, with certain recommendations, to the Secretary for Overseas Trade for investigation and pressing for early release of information so that exporters could be informed of the situation. The Committee strongly expressed the hope that when the Board of Trade were negotiating future Trade Agreements, they would consult the Group on the subject of live and bloodstock and that efforts would be made to give such stock a priority in trade lists.

Notification has since been received from the Secretary for Overseas Trade that these details have been forwarded to H.M. Embassy at Buenos Aires, asking the Minister (Commercial) to approach the Argentine authorities on the matter. H.M. Government have been pressing for the quick release of import licences, but they have been informed that until Argentina's sterling balances have recovered from their present low level, the implementation of Schedule 3 of the Agreement cannot yet be fully effected.

Quarantine and Veterinary Tests

Since the beginning of April last a further 17 quarantine periods have taken place when 312 head of livestock were accommodated in quarantine stations for ultimate shipment overseas. Figures are given below, showing countries to which stock was shipped with comparative figures for the corresponding period last year. The figure shown for stock shipped to South Africa is for shipments to the end of July, 1949, only, after which date animals have been shipped direct without entry into quarantine. There are at the moment some 84 head of stock in the London and Liverpool quarantine stations awaiting shipment to New Zealand, Australia and Eire:—

	April 1949 to Sept. 49	April 1948 to Sept. 1948
Australia	48	38
New Zealand	37	87
S. Africa, S.W. Africa and S. Rhodesia	80	191
U.S.A. (via Canada) and Canada	74	54
W. Indies	6	—
Eire	67	133
	312	503
Direct shipments:—		
U.S.A. (via Canada) and Canada	248	140
S. Rhodesia	Not available	68
	560	711

Despite the decrease in the total figure, which has to some extent been caused by the slowing down of shipments due to the dock strike, it is encouraging to note the rise in exports to the dollar area. From registrations received it is anticipated that after December 1st next shipments of cattle to Australia will be large.

Occasionally the Ministry receives complaints of loss of condition or of inadequate grooming of animals in quarantine stations. Actually, once animals have settled down in their new quarters in the stations, the great majority put on weight on the quarantine diet which long experience has shown to be both adequate and satisfactory. Where this improvement has not been observed there is reason to believe that in at least some instances it may be connected with the steaming up process to which some animals are subjected for show or sale purposes. Obviously it is impracticable to continue this practice in quarantine stations or on board ship and exporters are advised to put their animals on ordinary rations for as long as possible before sending them to the stations.

The complaint about inadequate grooming has probably arisen as the result of the effect of the dressings which are applied as a routine measure to all animals in quarantine stations. Importing countries require that animals must be free from external parasites and to ensure this they are dressed in the stations. Such dressings to be effective should remain on the coat for some time and they give it a dusty and, sometimes, ungroomed appearance. Every effort is made to remove all traces of these dressings before shipment, but this is not always possible, especially where several applications have to be made. It should be stated that grooming of stock in the stations is a daily routine procedure which is strictly adhered to.

The Ministry of Agriculture have notified that, as a result of representations made to the Venezuelan authorities, that country has now agreed to the direct importation of stock from Great Britain. Animals will require to enter quarantine before shipment, and shipping agents are asked to lodge quarantine applications with the Group in the normal way. It is hoped that every effort will be made by exporters in this country to open up a market there for British pedigree livestock.

The Canadian Department of Agriculture state that until further notice, cattle from England, Scotland and Ireland will not be subjected to the Johnin Test whilst undergoing quarantine in Canada.

It was considered opportune by the Executive that representation should be made to the delegates of overseas countries attending the International Veterinary Congress held in August to emphasise the desirability for uniformity in the application of veterinary tests. After consultation with members of the Ministry, it was decided that the matter should be taken up by them. Conditions of entry into Canada, South Africa, Australia and Argentina were discussed and it is hoped that some modification will be made.

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THE BRITISH PHARMACEUTICAL CODEX, 1949

The British Pharmaceutical Codex, 1949,* was published on October 17th, thirteen months after the Pharmacopoeia to which (says the *Pharmaceutical Journal*) it is more than ever complementary. Although it contains more words than its immediate predecessor, the fourth (1934) Codex, it is about 200 pages smaller; it is arranged in seven parts, an increase of three, two of which result from the segregation of the monographs on antisera and related products and on surgical catgut from the main part of the book, while the third, that on preparations of human blood, is entirely new. The last section is also notable in that the substances described are not given Latin titles. With each copy of the book there is a leaflet supplying the non-proprietary names under which many substances are included in the Codex, together, in some cases, with a selection of the more familiar brand names. This leaflet should be of great use in assisting readers of the Codex to interpret some of the newer and unfamiliar titles.

The preparation of the fifth Codex was begun in 1939, but was postponed for the duration of the war, and instead, the 1934 Codex was kept up to date (as far as possible) by the issue between 1940 and 1945 of seven supplements. By means of these, wartime advances in medicine and pharmacy were noted and the restrictions in the supply of drugs were accommodated. In 1945, preparation of the new Codex was resumed under the direction of the Codex Revision Committee and Mr. T. C. Denston, who had been appointed Editor on the death of Mr. C. E. Corfield in 1945. The Revision Committee was assisted by sub-committees which dealt with the action and uses of drugs, galenical standards, ligatures and sutures, pharmaceutical chemistry, pharmacognosy, pharmacy and surgical dressings, as well as by numerous specialists. Invaluable advice and help was also received from the British Pharmacopoeia Commission, Government departments and other organisations.

The British Pharmaceutical Codex, 1949, has been completely rewritten and rearranged and is now divided into seven parts. Part I contains the general monographs; Part II, antisera, vaccines and related products; Part III, preparations of human blood; Part IV, surgical ligatures and sutures; Part V, surgical dressings; Part VI, formulae; and Part VII, appendices.

* Pp. xxv + 1562. Price 63s. net. Inland postage, 1s. The Pharmaceutical Press, 17, Bloomsbury Square, London, W.C.1.

"INTERNATIONAL VETERINARY CONGRESS PRIZE"

From time to time, and in various journals, references under the above ambiguous heading have appeared to the award to which, in its current issue and in its account of the 86th annual meeting of the American Veterinary Medical Association held at Detroit in July, that Association's *Journal* applies the correct designation, *viz.*, "Twelfth International Veterinary Congress Prize." The history of the award was given briefly as follows by retiring President Hurt in presenting it, for this year, to Dr. Gerard Dikmans (of the B.A.I. Zoological Division) for his distinguished work in the field of veterinary parasitology. "Funds left over after the holding of the International Veterinary Congress in America, the only time it was held here, were placed in a fund, the income from which was to be used to give an annual award to men who have done some outstanding work along the lines of their profession."

At this meeting our recent welcome guest, Dr. C. P. Zepp, Sen., whose contribution to the London Congress of the N.V.M.A. appeared in our issue of October 8th, was installed as President, with the emblematic presentation of a gold key.

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MINISTRY OF AGRICULTURE NEWS SERVICE

Fertility in Farm Animals.—This is the time to take stock of the flocks and herds and plan for still greater output. A rising or improving state of nutrition before service is more favourable to fertility than a stationary or falling one. Flushing the ewes stimulates the reproductive organs, bringing the flock rapidly to the tup and increasing the number of twins. Food has a very great influence on fertility, as is seen when hill ewes are brought down to lowland pasture; and it is equally true of other farm animals. Various feeds may be used for flushing, but nothing is better than actively growing, succulent, mineral-rich green food, *e.g.*, fresh pasture. In present circumstances April lambing is early enough for most grassland flocks, and rain has come in time to give new life to the parched fields.

But it is not only sheep that we have to think about. For the past few years there has been a serious falling off in supplies of late summer milk. This year the August and September shortage has been accentuated by the severe drought, but it has been evident in other seasons not so affected. There are, of course, difficulties in the way of rapid readjustment of cow-calving dates, but with heifers the case is different. Indeed, so far as out-wintering heifers are concerned, earlier service, while the grass is still nutritious and the heifers have not lost bloom and condition, would make for fewer misses. Factors that lower nutrition and vitality, such as food shortage and bad weather, have a corresponding effect on fertility.

Animals, sometimes, may be too fat for breeding. Especially is this true of bulls and rams forced for show or sale purposes. It is well known that ewes should be in improving condition when they come to the ram, and the same is probably true of the ram himself. In many cases, however, he is losing condition all through tupping time, and it is reasonable to suppose that a little something as near akin as possible to the old-fashioned mixture of oats, bran and linseed cake, would help keep him up to his work. . . . Strict attention must be paid to the ram's feet. A lame ram at mating is a not uncommon cause of loss. To keep them sound a weekly run through the bluestone foot-bath is well worth while where foot rot is troublesome.

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Ragwort.—Ragwort was scheduled (along with thistles and docks) as a "noxious" weed under the Corn Production (Repeal) Act, 1921, and regulations, which are still in force, were made to enforce its control on agricultural land. There is not much doubt, however, that it is still on the increase in many areas. Ragwort is poisonous to stock, especially to cattle, and its coarse, unpalatable growth kills out and replaces much of the more useful herbage in the pasture, making it all the more desirable in these days that it should be kept under control.

The eradication of ragwort on grassland which is suitable for ploughing is simple. Ploughing up and putting through an arable rotation will get rid of it completely; and provided the land is in good heart when put back of grass, there is little chance of further trouble. Alternatively direct reseeding will give a vigorous sward which, with proper manuring and management, will give the ragwort no opportunity to re-establish itself.

On land that is not suitable for ploughing the successful control of ragwort depends in the long run on making the conditions in any particular pasture more suitable for the grass and clover and less suitable for the weed. On land in poor heart conditions are never suitable for the maximum growth of the better species, but the ragwort thrives because it will tolerate a low state of fertility. The only way to ensure permanent control, therefore, is to build up the fertility of the land sufficiently for the better grasses and clovers to flourish and crowd out the weeds.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

CASE VISITATION *versus* TREATMENT AT SURGERY

Sir,—I read Mr. Ambrose's interesting contribution to the discussion on the Organisation of Veterinary Clinical Practice with mixed feelings. As a layman and a dog breeder of many years' experience, it appears to me that the practice of taking animals to the surgery or clinic is open to a variety of objections from the *clients'* point of view. We are all too well aware of the dangers from infection, and the careful small-animal owner does his best to prevent his dog or cat coming into contact with hard pad, distemper and the like. However beautifully kept, a veterinary surgeon's waiting room is necessarily a fruitful source of contagion, as a dog may easily be suffering from (as yet) undiagnosed distemper or hard pad, or be "incubating" and equally infectious. I would never, myself, dream of taking a young dog to a veterinary surgery if I could possibly avoid it.

Another objection—again from the client's point of view—is that, if the animal is slightly "off colour," with the beginning of mild pyrexia, taking him from the warmth and comfort of his home and out to the clinic may well have a deleterious effect, if only from the change of temperature. Many owners might feel an obligation to take their animals to the surgery even if obviously not well, in view of the practitioner's plainly expressed preference for this course. The client is, after all, not in a position to decide whether his animal's ailment is of sufficient import as to necessitate a personal visit and it might not occur to him to find out.

I do feel that, though the practice followed by Mr. Ambrose has many obvious advantages from the veterinary surgeon's point of view, there is another aspect to be considered, to which I have ventured to draw attention.

Yours faithfully,

(Miss) Olwen Gwynne-Jones.

Mauldens, Ralph's Ride, Bracknell, Berks.

October 26th, 1949.

DISEASES OF ANIMALS ACTS, 1894 TO 1937, AND AGRICULTURE ACT, 1937 (PART IV).

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange*	Sheep Scab	Swine Fever
Oct. 1st to 15th, 1949	11	—	47	—	1	—
Corresponding period in—						
1948	5	—	1	—	4	1
1947	4	—	147	—	2	—
1946	3	—	—	—	—	5
Jan. 1st to Oct. 15th, 1949	177	13	455	—	34	5
Corresponding period in—						
1948	84	2	250	1	52	24
1947	91	103	1008	1	66	29
1946	72	33	—	7	66	331

Note.—The figures for the current year are approximate only.

* Excluding outbreaks in Army Horses.

Tuberculosis Order of 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period July 1st to September 30th, 1949, was 1,272.

Advertiser's Announcement

Willows Francis Pharmaceutical Products, Ltd.

An amalgamation has recently been completed between Willows, Francis, Butler & Thompson, Ltd., and the Pharmaceutical Manufacturing Co., Ltd., of The Laboratories, Pittville Circus Road, Cheltenham, and a new company, Messrs. Willows Francis Pharmaceutical Products, Ltd., has been formed and acquired both undertakings.

Both branches will continue to trade under their existing managements, but the London branch at 73, 75 & 80a, Shacklewell Lane, E.8, will trade under the name of the new company, and the Cheltenham branch will trade as Pharmaceutical Manufacturing Company (Proprietors: Willows Francis Pharmaceutical Products, Ltd.).